

REMARKS

Claims 30-39 and 67-89 are pending and are the subject of the outstanding office action.

Applicants wish to thank Examiners Kaufman, Eyler and Elliot for the telephonic interview on February 26, 2003 to discuss the objections and rejections raised in the office action.

Each of the objections and rejections set forth in the office action is addressed below.

Pursuant to the Examiner's request, the title of the invention has been amended, as shown herein.

The reference to Applicants' respective prior applications ("Related Applications") has also been amended on page 1 of the specification, pursuant to the Examiner's request.

Regarding the objection raised to claim 77 as being of improper dependent form, Applicants respectfully disagree. Claim 76, from which claim 77 depends, recites the effect in "one or more mammalian cells, while claim 77 further specifies that such cells are "one or more mammalian cancer cells". Accordingly, it is believed that claim 77 does not recite the same limitations as claim 76, and it is requested that this objection be withdrawn.

Formal drawings are being filed concurrently with this Response to correct the informalities noted by the draftsman. The Brief Description of the Drawings on page 7 of the specification has been amended to bring the figure numbering into conformance with the numbering used in the formal drawings.

The above-mentioned amendments to the specification and claims are illustrated on the attached pages entitled "Marked Up Version to Show Changes Made". For the Examiner's convenience, a clean copy of the now amended text in the specification and the now pending claims 30-39 and 67-89 is provided above.

Section 112 Rejections

Claims 67, 75, and dependent claims 68-74 and 76-84 were rejected under Section 112, second paragraph, as being indefinite. Applicants respectfully disagree that the term "specifically" introduces

ambiguity into the terms. Applicants have amended independent claims 67 and 75, as well as independent claims 85-89, to delete the term "specifically". This amendment, which is fully supported by the application, is being made to expedite the prosecution of this application and to place the claims in condition for immediate allowance; the amendment is not being made, and should not be interpreted, as acquiescence to the rejection. Applicants do not believe that the amendment narrows the scope of the claims in any manner.

Priority

In the office action, the Examiner asserts that the present application is not granted benefit of priority to its provisional application no. 60/059,288. The Examiner also asserts that for prior art purposes, the effective filing date of the present application is that of its provisional application no. 60/094,640. For the reasons below, the undersigned requests that these statements regarding priority be withdrawn.

Applicants respectfully submit that the issue of priority should be held in abeyance until such a time that the patent office has a consistent and appropriate standard for assessing utility and enablement in a patent application and in art references which the office intends to apply for prior art purposes. Applicants point out that the reference being applied by the Examiner in the Section 102(e) rejection (Emery et al., discussed below) discloses only certain sequence structure of the molecule called TR4. The inconsistency of patent office standards is clearly illustrated here - how can the disclosure of Emery et al. be deemed to satisfy requirements of utility and enablement and yet Applicants' provisional application 60/059,288 be found to lack specific utility? If the Examiner believes that Applicants' provisional application 60/059,288 fails to disclose specific utility, it is respectfully submitted that the disclosure of Emery et al. must be found to be similarly deficient.

Section 102 Rejections

Claims 67-77 were rejected under Section 102(e) as being anticipated by Emery et al. Applicants respectfully traverse this rejection.

The Emery et al. reference discloses certain sequence structure information for the TR4 molecule but fails to disclose any function or utility of the TR4 molecule. Emery et al. merely speculate what the function or activity TR4 might be, and the disclosure in Emery et al. relating to what TR4 may be used for or how it may be used is entirely prophetic and speculative. Contrary to the assertion in the office action (page 4), Emery et al. does not teach one skilled in the art what ligand(s) bind to TR4.

Emery et al. do not teach or suggest any DcR3 antibodies, such as claimed in the instant application, and withdrawal of the rejection is respectfully requested.

Respectfully submitted,

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By: _____



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Marked Up Version to Show Changes Made

In the Title of the Invention:

Please amend the Title as follows:

--- Antibodies to DcR3 Polypeptide, a TNFR Homolog ---

In the specification:

In the paragraph on page 1, lines 8-12, the text has been amended as follows:

---- RELATED APPLICATIONS

This application is a continuation application of Serial No. 09/157,289 filed September 18, 1998, now abandoned, which is a non-provisional application claiming priority under Section 119(e) to provisional application number 60/059,288 filed September 18, 1997 and to provisional application number 60/094,640 filed July 30, 1998, the contents of which are hereby incorporated by reference. ---

In the paragraph on page 7, lines 24-26, the text has been amended as follows:

--Figures 10A-D show[s] the results of assays to determine amplification of the DcR3 gene in various lung and colon tumors and in various colon tumor cell lines.---

In the claims:

Please amend claim 67 as follows:

67. (Amended) An isolated anti-DcR3 antibody which (a) [specifically] binds to a DcR3 polypeptide consisting of amino acids 1 to 215 or amino acids 1 to 300 of Fig. 1 (SEQ ID NO:1) and (b) inhibits binding of Fas ligand to the DcR3 polypeptide of (a).

Please amend claim 75 as follows:

75. (Amended) An isolated anti-DcR3 antagonist antibody which (a) [specifically] binds to a DcR3 polypeptide consisting of amino acids 1 to 215 or amino acids 1 to 300 of Fig. 1 (SEQ ID NO:1), (b) blocks binding of Fas ligand to the DcR3 polypeptide of (a), and (c) neutralizes inhibitory effect of the DcR3 polypeptide of (a) on Fas ligand activity in one or more mammalian cells.

Please amend claim 85 as follows:

85. (Amended) An isolated anti-DcR3 antibody which [specifically] binds to a DcR3 polypeptide comprising amino acids 1 to 215 of Fig. 1 (SEQ ID NO:1) and binds to the same DcR3 polypeptide epitope as the epitope to which the 4C4.1.4 monoclonal antibody produced by the hybridoma cell line deposited as ATCC accession number HB-12573 binds.

Please amend claim 86 as follows:

86. (Amended) An isolated anti-DcR3 antibody which [specifically] binds to a DcR3 polypeptide comprising amino acids 1 to 215 of Fig. 1 (SEQ ID NO:1) and binds to the same DcR3 polypeptide epitope as the epitope to which the 5C4.14.7 monoclonal antibody produced by the hybridoma cell line deposited as ATCC accession number HB-12574 binds.

Please amend claim 87 as follows:

87. (Amended) An isolated anti-DcR3 antibody which [specifically] binds to a DcR3 polypeptide comprising amino acids 1 to 215 of Fig. 1 (SEQ ID NO:1) and binds to the same DcR3 polypeptide epitope as the epitope to which the 11C5.2.8 monoclonal antibody produced by the hybridoma cell line deposited as ATCC accession number HB-12572 binds.

Please amend claim 88 as follows:

88. (Amended) An isolated anti-DcR3 antibody which [specifically] binds to a DcR3 polypeptide comprising amino acids 1 to 215 of Fig. 1 (SEQ ID NO:1) and binds to the same DcR3 polypeptide epitope as the epitope to which the 8D3.1.5 monoclonal antibody produced by the hybridoma cell line deposited as ATCC accession number HB-12571 binds.

Please amend claim 89 as follows:

89. (Amended) An isolated anti-DcR3 antibody which [specifically] binds to a DcR3 polypeptide comprising amino acids 1 to 215 of Fig. 1 (SEQ ID NO:1) and binds to the same DcR3 polypeptide epitope as the epitope to which the 4B7.1.1 monoclonal antibody produced by the hybridoma cell line deposited as ATCC accession number HB-12575 binds.